ACUTE TOXICITY SUMMARY

SODIUM HYDROXIDE

(caustic soda, caustic flake, white caustic, soda lye, lye, sodium hydrate)

CAS Registry Number: 1310-93-2

I. Acute Toxicity Summary (for a 1-hour exposure)

Inhalation Reference Exposure Level	**8 μg/m³**
Critical effect(s)	subjective complaints of eye, skin, and respiratory irritation in occupationally exposed workers
Hazard Index target(s)	Eyes; Skin; Respiratory System

II. Physical and Chemical Properties (HSDB, 1993)

Description	colorless solid
Molecular formula	NaOH
Molecular weight	40.01
Density	2.13 g/cm^3 at 25°C
Boiling point	1,390°C
Melting point	318.4°C
Vapor pressure	1 mm Hg @ 739°C
Flashpoint	not applicable
Explosive limits	not applicable
Solubility	soluble in water, alcohol and glycerine
Odor threshold	not applicable
Metabolites	not applicable
Conversion factor	not applicable (when dust)

III. Major Uses and Sources

Sodium hydroxide (NaOH) is produced primarily by the electrolysis of sodium chloride solutions and also from sodium carbonate. Sodium hydroxide is used in the manufacture of chemicals, rayon, soap and detergents, pulp and paper, petroleum products, cellophane, textiles and explosives, in etching and electroplating, in metal descaling, and in batteries.

IV. Acute Toxicity to Humans

Sodium hydroxide is a strong irritant and has a marked corrosive action on all body tissues regardless of the route of exposure (Reprotext, 1993). It is also more irritating than equivalent amounts of strong acid.

Controlled dermal exposures with concentrated sodium hydroxide have resulted in intercellular edema, erythema, decomposition of keratin material, and destruction of the epidermis (NIOSH, 1975). There may be a latency period between dermal contact and the onset of a sensation of irritation or burning. Exposure to sodium hydroxide mist may cause multiple small burns and temporary hair loss.

Sodium hydroxide in contact with the eyes can result in ulceration, perforation, and opacification of the cornea, leading to blindness (Grant, 1986; NIOSH, 1975).

Oral ingestion of sodium hydroxide can result in burns to the lips, tongue, oral mucosa, and esophagus (Medical Management, 1993). Sodium hydroxide has been implicated in the production of esophageal cancer at the site of esophageal strictures resulting from accidental ingestion (Appelqvist *et al*., 1980). These cancers are believed to be the consequence of scar formation and tissue destruction rather than from a direct carcinogenic effect of sodium hydroxide (NIOSH, 1975).

The effects of inhalation exposure to sodium hydroxide have not been reliably studied. Some cases of acute respiratory symptoms following exposure with nose and throat irritation, chest pains, and shortness of breath have been reported (NIOSH, 1974). In an unreferenced comment, Patty (1949) remarked that exposure to 2 mg /m³ NaOH in air is noticeably, but not excessively, irritating. Ott *et al*. (1977) stated that workers exposed to sodium hydroxide levels estimated to range from 0.5 to 2 mg/m³ time-weighted average (TWA) experienced nasal, skin, and, to a lesser extent, respiratory irritation. The duration of exposure prior to development of symptoms was not mentioned. Also, the 8-hour TWA concentrations are based on a one-time measurement. Workers exposed to 0.01 to 0.7 mg/m³ heated sodium hydroxide, in addition to other solvents, experienced upper respiratory tract irritation (Hervin and Cohen, 1973). Heating may increase the toxicity of sodium hydroxide (NRC, 1984).

Case reports exist in the literature of irreversible obstructive lung disease following chronic occupational exposure as well as after a one-time, high-level exposure to sodium hydroxide (Hansen and Isager, 1991; Rubin *et al*., 1992).

Predisposing Conditions for Sodium Hydroxide Toxicity

Medical: Persons with skin, eye or respiratory conditions may be more sensitive to the effects of sodium hydroxide (Reprotext, 1999). Persons with glaucoma should not work around mists or aerosols of sodium hydroxide since it can raise eye pressure (Reprotext, 1993).

Chemical: Persons exposed simultaneously to ammonium chloride, other irritants, or alkalis may be more sensitive to the effects of sodium hydroxide (Dluhos *et al*., 1969).

V. Acute Toxicity to Laboratory Animals

Application of sodium hydroxide to the skin of rats and mice has produced severe irritation leading to necrosis and death (NIOSH, 1975). Topical ocular application of sodium hydroxide in rabbits has resulted in ulceration, perforation, and corneal necrosis (NIOSH, 1975; Grant, 1986). Corneal opacification, vascularization, and an increase in intraocular pressure have also been observed. Species differences in the degree of irritancy and recovery after eye application have been noted (Grant, 1986). The eyes of monkeys are less sensitive to sodium hydroxide and recover more completely than rabbits' eyes.

In rats exposed by inhalation to an unknown concentration of sodium hydroxide produced from an aerosolized 40% solution for 30 minutes twice daily for 2.5 months, lung examination revealed alveolar wall thickening with cell proliferation and congestion (Dluhos *et al*., 1969). Ulceration and flattening of the bronchial epithelium and proliferation of lymphadenoid tissue were also reported. Undescribed, isolated tumors were observed in 3 of 10 animals. In another study, inhalation exposure twice weekly for one month to an aerosol produced from a 40% sodium hydroxide solution resulted in the deaths of all 27 rats, predominantly from bronchopneumonia (Vyskocil *et al*., 1966). Exposure to an aerosol produced from a 20% solution of sodium hydroxide produced dilatation and destruction of alveolar septae. Although no effects were observed in the group exposed to a 10% solution, in rats exposed to aerosolized 5% sodium hydroxide, bronchial dilatation and mucus membrane degeneration were observed, which suggest a poor dose-response relationship in this study.

VI. Reproductive or Developmental Effects

No studies are available regarding the reproductive or developmental effects of sodium hydroxide in humans.

Sodium hydroxide injected into the amniotic fluid of rats at 0.001 M on day 13 of gestation was not teratogenic but was slightly embryotoxic (Dostal, 1973).

VII. Derivation of Acute Reference Exposure Level and Other Severity Levels (for a 1-hour exposure)

Reference Exposure Level (protective against mild adverse effects): 0.008 mg/m^3 (8 μg/m^3)

Study	Ott *et al*., 1977
Study population	291 workers in sodium hydroxide production
Exposure methods	occupational exposure
Critical effects	subjective reports of mild to moderate-severe irritation of the eyes and skin; mild respiratory irritation
LOAEL	0.5 mg/m^3
NOAEL	not reported
Exposure duration	8-hour time-weighted average

Extrapolation to 1 hour	not used
LOAEL uncertainty factor	6
Interspecies uncertainty factor	1
Intraspecies uncertainty factor	10
Cumulative uncertainty factor	60
Reference Exposure Level	0.008 mg/m³ (8 μg/m³)

Uncertainty factors were applied to the lowest concentration at which symptoms were reported (0.5 mg/m³). The reported irritation was mild to moderate-severe, which indicates that the irritation was beyond mild irritation although it was below severe classification. Because sodium hydroxide aerosols can readily undergo reaction with carbon dioxide to form sodium carbonate, a standard for sodium carbonate should also be developed (Cooper *et al*., 1979).

Level Protective Against Severe Adverse Effects

No recommendation is made due to the limitations of the database.

In an unreferenced comment, Patty (1949) stated that exposure to 2 mg/m³ sodium hydroxide in air would cause noticeable, but not excessive respiratory irritation. Exposure to sodium hydroxide estimated to be as high as 2 mg/m³ TWA caused nasal and skin irritation, especially in areas of the plant where temperatures were higher (Ott *et al*., 1977).

The NRC (1984) used their expert judgment in determining an EEGL of 2 mg/m³, therefore it does not follow OEHHA's methodology. No margin of safety was applied in the derivation of the 1-hour EEGL.

Level Protective Against Life-threatening Effects

No recommendation is made due to the limitations of the database.

NIOSH (1995) reports an IDLH of 10 mg/m³. It is based on Ott *et al*. (1977). Workers exposed to 2 to 8 mg/m³ "caustic dust" experienced irritation of the respiratory system. NIOSH states that "This may be a conservative value due to the lack of relevant acute toxicity data for workers exposed to concentrations above 8 mg/m³."

VIII. References

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